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Original article

Elevated metabolic rate during passive stretching is not a sufficient aerobic warm-up

Arnold G. Nelson [a,*], Joke Kokkonen [b]

[a] *School of Kinesiology, Louisiana State University, Baton Rouge, LA 70803, USA*
[b] *Exercise & Sport Science Department, Brigham Young University-Hawaii, Laie, HI 96762, USA*

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Abstract

Purpose: The purpose of this study was to determine the extent that a static stretching program could increase heart rate (HR) and oxygen consumption (VO_2), and if the increases were sufficient to serve as a warm-up for aerobic activity.
Methods: The HR and VO_2 of 15 male and 16 female college students were measured after either 12 min of passive static stretching (SS), or 12 min pseudo-stretching (PS), which consisted of moving through the stretching positions without placing the muscles on stretch. Four different lower body stretches were used with each stretch held for 30 s before the participant moved to a different position, with the circuit being repeated four times. VO_2 was determined by averaging breath-by-breath measures over the total 12 min. HR was obtained every 30 s and the 24 values were averaged. Warm-up benefit was determined from the O_2 deficit accrued during 7-min cycling at 60% VO_{2max}.
Results: HR (beats/min, mean ± SD) for SS (84 ± 11) was a significant ($p < 0.05$) 9% greater than PS (78 ± 12). Similarly, VO_2 (mL/min, mean ± SD) for SS (0.53 ± 0.13) was a significant 44% greater than PS (0.38 ± 0.11). The O_2 deficit (L, mean ± SD) for SS (0.64 ± 1.54) was not different from PS (0.72 ± 1.61).
Conclusion: These data indicate that passive static stretching increases both HR and VO_2, indicating that metabolic activity can be increased without muscle activation. The magnitude of the increases, however, is not sufficient to elicit a warm-up effect.

Keywords: Acute stretching; Heart rate; Metabolic activity; Oxygen consumption; O_2 deficit; Passive stretching

1. Introduction

Historically, it is widely conjectured that stretching will promote better performances and reduce the incidence of injury.[1,2] Consequently, exercises designed to enhance flexibility are regularly included in the pre-event warm-up activities of many athletes.[3] Notwithstanding the widespread acceptance and use of stretching exercises as a major component of pre-event warm-up, the purported benefits of stretching upon performance have come into question. Over the past decade, research has established an adverse effect of acute static stretching upon various different maximal performances. Pre-event stretching has demonstrated an inhibitory effect upon maximal force or torque production,[4–7] vertical jump performance,[8–10] running speed,[11–13] and muscular endurance.[14,15] Thus, it would appear that stretching is contraindicated as a warm-up modality for anaerobic activities.

Even though stretching may be contraindicated for anaerobic activities, the literature suggests that stretching may not be detrimental for aerobic activities. First, it is well established that activation of skeletal muscle mechanoreceptors result

* Corresponding author.
E-mail address: anelso@lsu.edu (A.G. Nelson)
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in increased heart rate (HR). This is due to both a decrease in parasympathetic drive, as well as an increase in sympathetic drive.[16] While static and dynamic muscle contractions are the primary method by which mechanoreceptors are activated, passive stretching can also cause a cardiovascular response. Using passive stretch, Stebbins et al.[17] noted an increase in HR that was approximately one-half of that observed during muscle contractions. They found that this cardiovascular response resulted from an initial increase in sympathetic drive, and a succeeding decrease in parasympathetic influence. Subsequent studies in humans have confirmed this relationship between passive stretch and HR. For example, Gladwell and associates[18,19] found that sustained passive stretch can cause a significant increase in HR. Their data suggested that the elevations in HR were due to small fiber muscle mechanoreceptors inhibiting cardiac vagal activity. Also, Cui et al.[20] stretched calf muscles 25 times using cycles consisting of 5-s stretch and 15−25-s relaxation. They found that passive stretch of the calf muscles induced transient and significant increases in both HR and muscle sympathetic nerve activity. In addition to increasing HR, several studies using either cell culture or isolated animal muscles suggested that the passive static stretch of muscles could result in elevated metabolic activity. In 1932, Feng[21] found that passively stretched muscles exhibited increased heat production and oxygen consumption. Later research corroborated his findings, with Clinch[22] reporting increased heat production, and Whalen et al.[23] and Barnes[24] reporting increased oxygen consumption. In other related studies, passive stretch induced increased carbon dioxide production,[25] increased glycogen breakdown,[26] and increased lactic acid production.[24] Finally, Nelson and associates[27] showed that passive stretching decreased blood glucose levels.

In his reviews on warm-up practices, Bishop[28,29] reported several studies that linked warm-up to improved aerobic performance. Largely, the improved aerobic performance due to warm-up activities was proposed to act by speeding up the limiting reaction(s) associated with oxidative phosphorylation. In addition, any rise in muscle temperature could cause both increased in muscle vasodilation and a rightward shift of the oxyhemoglobin dissociation curve which would facilitate O_2 delivery during the transition to exercise.[30] These changes would allow an individual to begin with an elevated HR and oxygen consumption the subsequent task, and thus reach a higher level of aerobic metabolism more quickly.[31] This is hypothesized to reduce the initial O_2 deficit and enable the anaerobic system to contribute to energy supply over period of time.[32] Moreover, the reduction in O_2 deficit is more likely to happen for exercise above the lactate threshold.[30] Therefore, the purpose of this study was twofold. The first purpose was to determine if a stretching program consisting of four passive stretches would increase not only a person's HR but also his/her oxygen consumption. The second purpose was to determine if any increase in HR or oxygen consumption was sufficient to alter the O_2 deficit during the first 7 min of a subsequent aerobic exercise.

2. Methods

2.1. Participants

Participants consisted of 15 male and 16 female college students who were physically inactive (Table 1). Informed written and verbal consent was obtained from each participant prior to taking part in the experiment, and the appropriate institutional human participants review committee approved the study. The participants were not allowed to see the results until the study was completed.

2.2. Study overview

Participants made three visits to the laboratory. During the first visit the participants' stationary cycling peak oxygen consumption (VO_{2peak}) was determined using a protocol published previously.[33] On the two subsequent visits, each person's oxygen consumption and HR were recorded for three different levels of activity (i.e., quiet sitting, stretching or pseudo-stretching, and cycling at approximately 65%−70% VO_{2peak}). For the quiet sitting (QS) condition, each person rested quietly in a chair for 10 min. After resting for 10 min, each person did a regimen of either 12 min of passive static stretching (SS) or pseudo-stretching (PS). Two minutes after finishing the stretch treatment, the participants cycled for 7 min at a workload that approximated 65%−70% of their VO_{2peak}. During all conditions (QS, PS, SS, exercise) expired gases were monitored continuously using a Sensor Medics Vita Max breath-by-breath system (CareFusion Corporation, 3750 Torrey View Court, San Diego, CA, USA). Additionally, HR was monitored continuously via a POLAR® HR monitor (Polar Electro, Kempele, Finland) and recorded at 30-s intervals. The order of PS and SS was balanced across the days two and three, and minimum of 24 h and maximum of 72 h separated these two test days.

2.3. Stretching protocol

The SS program consisted of four different passive static stretching activities. The first stretching exercise was a sit-and-reach. The participant sat on the floor with the legs extended and the head and trunk were lowered toward the knees. The second activity was the lotus or butterfly stretch. Here the participant sat on the floor in the lotus position and then the head and trunk were lowered toward to the floor. For the third activity a standing half lotus was performed. While standing with one foot flat on the floor, the participant placed the

Table 1
Participant descriptive data.

Gender	Age (year)	Mass (kg)	Height (cm)	VO_{2max} (mL/kg/min)
Female ($n = 16$)	21 ± 2	68 ± 12	166 ± 6	32.6 ± 5.1
Male ($n = 15$)	24 ± 2	78 ± 13	178 ± 5	39.7 ± 5.1

Values are mean ± SD.

opposite leg in a lotus position upon a table. The head and trunk were then lowered toward the foot of the leg resting upon the table. The fourth and final exercise was a quadriceps stretch. For this stretch, the participant stood with their back to a table with the knee flexed so that the dorsal side of one foot lay upon the table. From this position, the flexed leg and the opposite shoulder were pushed toward the table. For each stretch, an experimenter pushed the participant in the aforementioned directions until the participant acknowledged that a stretch near their pain tolerance was felt in the stretched leg(s). This stretch was then maintained on the musculature for 30 s. After which the participant relaxed for 15 s, and then assumed either the position for opposite leg (stretches 3 and 4) or next position following the order listed above. This stretching circuit was repeated a total four times.

For the PS condition, the participant assumed each position for 45 s without placing any tension upon the musculature. After 45 s, either the position for opposite leg (stretches 3 and 4) or next position was assumed, and, again, the above circuit was repeated four times.

2.4. 65%−70% VO_{2peak} test

On days two and three each person did a 65%−70% VO_{2peak} test which commenced 2 min after completing either SS or PS. Prior to the person sitting on the cycle ergometer, a member of the research staff set the ergometer resistance to a level calculated (to the nearest 30 W) to achieve 65%−70% of the participant's VO_{2peak}. The participant then sat on the ergometer and immediately started to pedal at a 90 rpm cadence. The participant then cycled continuously at this workload for 7 min. As mentioned previously, breath-by-breath expired gases were obtained during this test, and used to calculated oxygen consumption, carbon dioxide production and respiratory exchange ratio (RER). In addition, HR was recorded every 30 s.

The total liters of oxygen consumed during the 7-min exercise were determined by calculating the area under the VO_2: time curve using the trapezoid rule.[34] In addition, the total energy used as well as the total energy derived from carbohydrate (CHO) and lipid oxidation for were calculated for each breath by multiplying VO_2 (L/min) and the respective energy expenditures (total, CHO, and lipid; kJ/min). The total, CHO, and lipid energy expenditures were obtained from the RER using the formulae of Lusk.[35]

Oxygen deficit following either PS or SS was calculated as the difference between the expected and actual oxygen consumed during the 7-min exercise. Actual oxygen consumed was calculated by using the trapezoidal rule,[34] and expected oxygen consumption was calculated from the prediction equations published by the American College of Sports Medicine.[36]

2.5. Data analysis

The dependent variables analyzed were HR, VO_2, and energy derived from CHO and lipid oxidation. For HR, the value analyzed for PS and SS was the average of the 24 30-s HRs recorded during each test. A paired t test was used to determine if a difference existed between the PS and SS dependent variables (i.e., HR, average VO_2 (mL/min), total CHO consumed, and O_2 deficit). An additional paired t test was used to determine whether or not an order effect for treatments was present (e.g., the HR and VO_2 results of the PS and SS treatments were collapsed across days). The level of significance was set at $p < 0.05$, and was adjusted to cover for multiple comparison (p value divided by number of comparisons). Hence, for significance at the 0.05 level to occur, the t score was actually tested at the $p < 0.0125$ level.

In addition, both the HR and total oxygen consumed for each 30-s period for PS and SS over the length of the stretch treatment were compared using a two-way ANOVA (stretch treatment × time) with repeated measures. When appropriate, Bonferroni t tests were used for *post-hoc* analysis (Microsoft Excel, Microsoft, Remond, WA, USA).

3. Results

3.1. Order (or day) effects

There were no significant differences found between the 2 days for either HR ($p = 0.56$) or average VO_2 ($p = 0.80$).

3.2. HR and VO_2

The changes in HR for PS and SS over the length of the stretch treatment are presented in Fig. 1. The main effect for stretch treatment (PS *vs.* SS) was significantly different ($F(1, 58) = 5.14$, $p < 0.027$) with the SS showing a higher HR. The main effect for time was also significant ($F(1, 23) = 8.98$, $p < 0.0031$). Additionally, a significant interaction ($F(23, 1334) = 2.84$, $p < 0.0001$) between stretch treatment and time was also found. As seen in Fig. 1, HR fluctuated during each treatment, and these changes appear to coincide

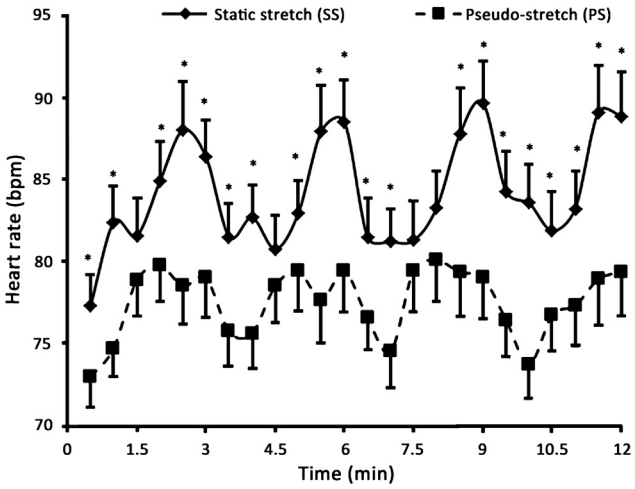

Fig. 1. Heart rate at each 30 s interval during the two stretching treatments. * indicates an SS value significantly greater than the corresponding PS value ($p < 0.05$).

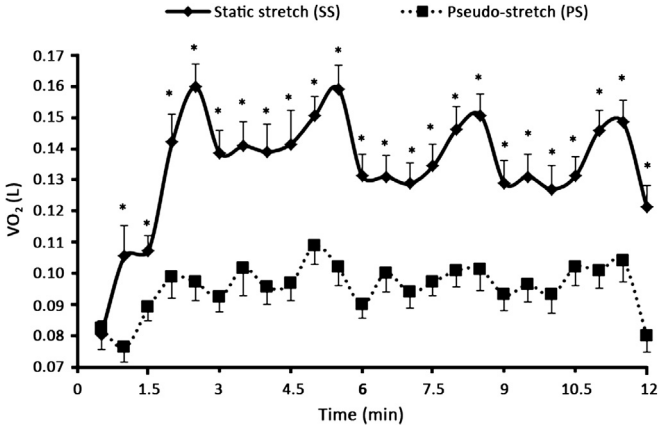

Fig. 2. Total oxygen consumed over the course of each 30-s interval during each stretch treatment. * indicates an SS value significantly greater than the corresponding PS value ($p < 0.05$).

with the changes in body position and the resumed application of each treatment. Nevertheless, *post-hoc* analyses showed that the PS HRs were significant lower than the SS HRs except at the 1.5, 4.5, 7.5, and 8 min time points. Finally the average HR during PS (78 ± 12 beats/min) was significantly lower ($p < 0.0001$) than the average HR during SS (84 ± 11 beats/min). Both of the average HR for PS and SS, however, were significantly greater than the average HR recorded during the 10 min of quiet sitting (70 ± 11 beats/min).

The changes in VO_2 for PS and SS over the length of the stretch treatment are presented in Fig. 2. The main effect for stretch treatment (PS *vs.* SS) was significantly different ($F(1, 58) = 25.81$, $p < 0.0001$) with the SS showing a higher VO_2. The main effect for time was also significant ($F(1, 23) = 18.5$, $p < 0.0001$). Additionally, a significant interaction ($F(23, 1334) = 4.97$, $p < 0.0001$) between stretch treatment and time was also found, and *post-hoc* analyses showed that the PS VO_2s were significant lower than the SS VO_2s except at the 0.5 min time point. Finally the average VO_2 during PS (0.38 ± 0.11 mL/min) was significantly lower ($p < 0.0001$) than the average VO_2 during SS (0.53 ± 0.13 mL/min). Both of the average VO_2 for PS and SS, however, were significantly greater than the average VO_2 recorded during the 10 min of quiet sitting (0.23 ± 0.06 mL/min).

3.3. Total kJ consumed

The kJ (total, CHO, fat) consumed during the PS and SS treatments are presented in Table 2. The average PS total energy consumption over the 12 min treatment was 28% lower than the SS total energy consumption, and this difference was statistically different ($p < 0.05$). Comparison between PS and SS for both CHO kJ and fat kJ consumption were similar to the total kJ consumption. On average, the PS CHO kJ was statistically ($p < 0.05$) lower (19%) than the SS CHO kJ, and PS fat kJ was significantly ($p < 0.05$) less (22%) than SS fat kJ. On the other hand, the relative amount of total energy consumed as CHO for PS (56.8%) and relative amount of total energy consumed as CHO for SS (56.6%) were not statistically different ($p = 0.96$).

Table 2
The average total, CHO, and fat consumption and O_2 deficit between the static stretch (SS) and pseudo-stretch (PS).

Variable	PS	SS
Total energy consumed (kJ)	93.9 ± 26.7	131.7 ± 33.3[a]
Total carbohydrate consumed (kJ)	52.9 ± 18.9	75.7 ± 32.9[a]
Total fat consumed (kJ)	41.1 ± 19.9	56.1 ± 21.9[a]
O_2 deficit (L)	0.72 ± 1.61	0.64 ± 1.54

Values are mean ± SD.
[a] Indicates an SS value that is significantly greater than PS ($p < 0.05$).

3.4. O_2 deficit

The O_2 deficit (L) for PS and SS are presented in Table 2. On average, the PS O_2 deficit was 18% greater than the SS O_2 deficit, but, statistically, PS and SS were not statistically different ($p = 0.84$).

4. Discussion

4.1. Stretching effects on HR and VO_2

A major purpose of this study was to determine if a program of four passive stretches would increase not only a person's HR, but also one's VO_2. An additional purpose was to determine if any differential increases in HR and VO_2, would translate into an adequate warm-up, demonstrated as a reduction in O_2 deficit. The above data indicate that the passive static stretching regimen used increases both HR, and resting VO_2 above and beyond that which is seen during either quiet sitting or moving between the four different body positions. These findings indicate that the previously demonstrated *in vitro* relationships between stretching and metabolism have correlates with *in situ* situations. This indicates that when a person performs assisted stretching, it cannot be assumed that muscle stretch induced and maintained by any outside forces is a passive muscle action, and therefore, a metabolically inert event.

This finding that "passive" exercise does not imply muscle inactivity is not new. In 2003, Bell et al.[37] recorded leg muscle activity via electromyography (EMG) during 5 min of passive exercise on either a tandem bicycle or a chair apparatus with ropes and weights. In both types of passive movement, noticeable levels of muscle activation were reported, demonstrating that the leg muscles were not quiescent during passive movements. Noticeable EMG responses to passive static stretches have also been reported by other researchers. For example, using surface electrodes, Condon and Hutton[38] reported EMG activity in a statically stretched soleus. The EMG activity during the static stretch, however, was less than that seen when using either agonist contraction or hold-relax techniques. In two studies, McHugh and associates[39,40] recorded hamstring surface electrode EMG during passive straight leg raises. Both studies saw an increase in hamstring EMG that was as much as 3% of that elicited during a maximum voluntary contraction. Interestingly, Etnyre and

Abraham[41] using implanted wire electrodes also found an increase in EMG activity. Contrary to data reviewed above, their reported increases in EMG were not observed in the stretched muscle, but rather in the antagonist muscle. This is a novel finding to our understanding and we are a loss to explain this phenomenon. Nevertheless, regardless of where the increase in EMG is originating, these studies show that passive static stretching results in some type of muscle activity, and this muscle activity could be the source of a significant, albeit small, increase in VO_2.

The increased VO_2 noted in our current investigation could be related to alterations in Ca^{2+} kinetics. Armstrong et al.[42] reported an increase in Ca^{2+} influx from extracellular spaces into the cells of isolated rat soleus muscles undergoing static stretching. Increased cytoplasmic Ca^{2+} can lead to an increased mitochondrial Ca^{2+} concentration. Increased mitochondrial Ca^{2+} concentration in turn results in an upregulation of the rate limiting enzymes found in the oxidative metabolic pathways,[43] and an accelerated oxidative pathway would lead to an enhanced VO_2. Furthermore, Ca^{2+} which is released must be returned to the sarcoplasmic reticulum which requires the utilization of ATP. This increase in ATP utilization may partially explain the need for an increase in metabolic rate. Interestingly, a chronic stretch study by Pattullo et al.[44] correlates with the aforementioned supposition that alterations in Ca^{2+} kinetics could be the mechanism behind the increase in VO_2. In their investigation, Pattullo and colleagues[44] placed the primarily fast glycolytic rabbit tibialis anterior muscle under chronic stretch. Following 5 weeks of this stimulus, the number of slow oxidative muscle fibers had increased fivefold. A fast-to-slow fiber transition is related to both increased oxidative work and Ca^{2+} concentrations.[44,45]

While increased HR and VO_2 must occur before one can consider any activity capable of including a training effect, the results of this study do not suggest that stretching exercises alone could either supplant or enhance existing training programs designed to lead to cardiovascular adaptations. The results of this study, especially when combined with the findings of Kokkonen et al.,[46] however, suggest that a stretching program alone may benefit individuals who have very low physical fitness or who are mobility impaired. The average VO_2 during the static stretching was of a magnitude similar to the estimated VO_2 for walking 1.5 mph. This suggests that several static stretching sessions could be used as an initial or adjunct exercise program for patients with low physical capacities (i.e., functional classes III & IV). The stretching sessions could initiate improvements in both the skeletal muscles as well as the cardiovascular system, especially if further studies are able to identify an optimum stretching dose. Additionally, the increased carbohydrate utilization supports the findings of Nelson et al.,[27] who showed that a 20-min stretching program significantly decreased blood glucose concentrations in an at-risk population. Thus, it would appear that exposes muscles on to short bouts of stretch has beneficial effects. Granted, this study did not exam severely challenged individuals, and so one cannot assume that the magnitude of change would be similar. Moreover, the stretching program would most likely need to be modified, because a physically challenged person may not be able to perform the exact same stretching regimen. Nevertheless, the possibility of a benefit exists and is worthy of further study. This study could serve as a springboard for further investigation into the use of passive static stretching alone as a modality to improve the physical work capacity of the very unfit.

4.2. Stretching as aerobic exercise warm-up

Classically, it has been proposed that warm up may improve aerobic performances of lasting less than 10 min by elevating HR and VO_2, thus decreasing the initial O_2 deficit.[28,29] An elevated baseline VO_2 allows for less of the initial work will be completed anaerobically, leaving more of the anaerobic capacity for the end of the task. This warm-up benefit may result from either increased oxygen delivery to the muscles via a rightward shift in the oxygen: hemoglobin dissociation curve, vasodilation of muscle blood vessels, or acceleration of the rate-limiting reactions of oxidative phosphorylation. Since, as mentioned above, stretching has the potential to raise HR and VO_2, the second research goal was to examine differences in O_2 deficit following the stretch treatments. In this research, the stretching regimen did significantly raise HR and VO_2, but the increases were not sufficient to alter the O_2 deficit. As mentioned above, the increase in average VO_2 during the static stretching was of a magnitude similar to the estimated VO_2 for walking 1.5 mph. As categorized by Bishop,[28,29] warm-up activities that improved performance raise the VO_2 to much higher levels. In fact, Gutin et al.[47] reported that a warm-up exercise equal to 30% VO_{2max} had no effect on aerobic exercise performance. The average VO_2 achieved during SS was equal to approximately 20% of the participants' VO_{2max}, thus it should not be surprising SS alone did not provide a significant warm-up. Moreover, the cycling activity commenced 3−5-min post stretching, and thus, it is possible that the VO_2 returned closer to the resting baseline in that intervening period and was actually lower than 20%. Obviously, there are other activities as easy to perform as stretching that would elevate the HR and VO_2 to a higher degree, and so, stretching solely for warm-up cannot be supported.

5. Conclusion

In summary, this study's data indicate that a passive static stretching program can increase HR, and resting VO_2 above and beyond that seen during either resting or moving between four different body positions. Thus, it is possible that regularly performing stretching exercises can provide more physical benefits than just an increased joint range of motion. It appears possible that passive static stretching may be an additional low intensity activity that severely physically limited individuals can use to initiate improvements in physical function. Furthermore, this investigation adds credence to previously published data demonstrating the passive static stretching does not suggest a lack of muscular activity and does have a metabolic cost.

References

1. Shellock FG, Prentice WE. Warming-up and stretching for improved physical performance and prevention of sports-related injuries. *Sports Med* 1985;**2**:267−78.
2. Smith CA. The warm-up procedure: to stretch or not to stretch. A brief review. *J Orthop Sports Phys Ther* 1994;**19**:12−7.
3. Jeffreys I. Warm-up and stretching. In: Baechle TR, Earle RW, editors. *Essentials of strength training and conditioning*. 3rd ed. Champaign, IL: Human Kinetics; 2008. p. 295−324.
4. Kokkonen J, Nelson AG, Cornwell A. Acute muscle stretching inhibits maximal strength performance. *Res Q Exerc Sport* 1998;**69**:411−5.
5. Fowles JR, Sale DG, MacDougall JD. Reduced strength after passive stretch of the human plantarflexors. *J Appl Physiol* 2000;**89**:1179−88.
6. Winchester JB, Nelson AG, Kokkonen J. A single 30-s stretch is sufficient to inhibit maximal voluntary strength. *Res Q Exerc Sport* 2009;**80**:257−61.
7. Costa PB, Ryan ED, Herda TJ, DeFreitas JM, Beck TW, Cramer JT. Effects of stretching on peak torque and the H:Q ratio. *Int J Sports Med* 2009;**30**:60−5.
8. Behm DG, Kibele A. Effects of differing intensities of static stretching on jump performance. *Eur J Appl Physiol* 2007;**101**:587−94.
9. Church JB, Wiggins MS, Moode FM, Crist R. Effect of warm-up and flexibility treatments on vertical jump performance. *J Strength Cond Res* 2001;**15**:332−6.
10. Cornwell A, Nelson AG, Heise GD, Sidaway B. The acute effects of passive muscle stretching on vertical jump performance. *J Hum Move Stud* 2001;**40**:307−24.
11. Nelson AG, Driscoll NM, Landin DK, Young MA, Schexnayder IC. Acute effects of passive muscle stretching on sprint performance. *J Sports Sci* 2005;**23**:449−54.
12. Winchester JB, Nelson AG, Landin D, Young MA, Schexnayder IC. Static stretching impairs sprint performance in collegiate track and field athletes. *J Strength Cond Res* 2008;**22**:13−9.
13. Beckett JR, Schneiker KT, Wallman KE, Dawson BT, Guelfi KJ. Effects of static stretching on repeated sprint and change of direction performance. *Med Sci Sports Exerc* 2009;**41**:444−50.
14. Nelson AG, Kokkonen J, Arnall DA. Acute muscle stretching inhibits muscle strength endurance performance. *J Strength Cond Res* 2005;**19**:338−43.
15. Franco BL, Signorelli GR, Trajano GS, de Oliveira CG. Acute effects of different stretching exercises on muscular endurance. *J Strength Cond Res* 2008;**22**:1832−7.
16. Murphy MN, Mizuno M, Mitchell JH, Smith SA. Cardiovascular regulation by skeletal muscle reflexes in health and disease. *Am J Physiol Heart Circ Physiol* 2011;**301**:H1191−204.
17. Stebbins CL, Brown B, Levin D, Longhurst JC. Reflex effect of skeletal muscle mechanoreceptor stimulation on the cardiovascular system. *J Appl Physiol* 1988;**65**:1539−47.
18. Gladwell VF, Coote JH. Heart rate at the onset of muscle contraction and during passive muscle stretch in humans: a role for mechanoreceptors. *J Physiol* 2002;**540**(Pt 3):1095−102.
19. Gladwell VF, Fletcher J, Patel N, Elvidge LJ, Lloyd D, Chowdhary S, et al. The influence of small fibre muscle mechanoreceptors on the cardiac vagus in humans. *J Physiol* 2005;**567**(Pt 2):713−21.
20. Cui J, Blaha C, Moradkhan R, Gray KS, Sinoway LI. Muscle sympathetic nerve activity responses to dynamic passive muscle stretch in humans. *J Physiol* 2006;**576**(Pt 2):625−34.
21. Feng TP. The effect of length on the resting metabolism of muscle. *J Physiol* 1932;**74**:441−54.
22. Clinch NF. On the increase in rate of heat production caused by stretch in frog's skeletal muscle. *J Physiol* 1968;**196**:397−414.
23. Whalen WJ, Collins LC, Berry H. Effect of stretch on O_2 consumption of resting and active frog sartorius muscle. *Am J Physiol* 1962;**203**:731−4.
24. Barnes WS. Respiration and lactate production in isolated frog skeletal muscle: effects of passive stretch. *Comp Biochem Physiol A Comp Physiol* 1987;**86**:229−32.
25. Eddy NB, Downs AW. Extensibility of muscle: the production of carbon dioxide by a muscle when it is made to support a weight. *Am J Physiol* 1921;**56**:188−95.
26. Barnes WS, Worrell GA. Glycogen utilization in isolated frog muscle: an effect of passive mechanical stretch. *Comp Biochem Physiol A Comp Physiol* 1985;**81**:243−6.
27. Nelson AG, Kokkonen J, Arnall DA. Twenty minutes of passive stretching lowers glucose levels in an at-risk population: an experimental study. *J Physiother* 2011;**57**:173−8.
28. Bishop D. Warm up I: potential mechanisms and the effects of passive warm up on exercise performance. *Sports Med* 2003;**33**:439−54.
29. Bishop D. Warm up II: performance changes following active warm up and how to structure the warm up. *Sports Med* 2003;**33**:483−98.
30. Koga S, Shiojiri T, Kondo N, Barstow TJ. Effect of increased muscle temperature on oxygen uptake kinetics during exercise. *J Appl Physiol* 1997;**83**:1333−8.
31. Gutin B, Stewart K, Lewis S, Kruper J. Oxygen consumption in the first stages of strenuous work as a function of prior exercise. *J Sports Med Phys Fitness* 1976;**16**:60−5.
32. Stewart IB, Sleivert GG. The effect of warm-up intensity on range of motion and anaerobic performance. *J Orthop Sports Phys Ther* 1998;**27**:154−61.
33. Nelson AG, Day R, Glickman-Weiss EL, Hegsted M, Kokkonen J, Sampson B. Creatine supplementation alters the response to a graded cycle ergometer test. *Eur J Appl Physiol* 2000;**83**:89−94.
34. Gibaldi M, Perrier D. *Pharmacokinetics*. New York: Dekker; 1982. p. 445−7.
35. Lusk G. Animal calorimetry. Twenty-fourth paper. Analysis of the oxidation of mixtures of carbohydrate and fat. A correction. *J Biol Chem* 1924;**59**:41−2.
36. American College of Sports Medicine. *ACSM guidelines for exercise testing and prescription*. 6th ed. Philadelphia: Lippincott Williams and Wilkins; 2000. p. 303.
37. Bell HJ, Ramsaroop DM, Duffin J. The respiratory effects of two modes of passive exercise. *Eur J Appl Physiol* 2003;**88**:544−52.
38. Condon SM, Hutton RS. Soleus muscle electromyographic activity and ankle dorsiflexion range of motion during four stretching procedures. *Phys Ther* 1987;**67**:24−30.
39. McHugh MP, Kremenic IJ, Fox MB, Gleim GW. The role of mechanical and neural restraints to joint range of motion during passive stretch. *Med Sci Sports Exerc* 1998;**30**:928−32.
40. McHugh MP, Magnusson SP, Gleim GW, Nicholas JA. Viscoelastic stress relaxation in human skeletal muscle. *Med Sci Sports Exerc* 1992;**24**:1375−82.
41. Etnyre BR, Abraham LD. Antagonist muscle activity during stretching: a paradox re-assessed. *Med Sci Sports Exerc* 1988;**20**:285−9.
42. Armstrong RB, Duan C, Delp MD, Hayes DA, Glenn GM, Allen GD. Elevations in rat soleus muscle $[Ca^{2+}]$ with passive stretch. *J Appl Physiol* 1993;**74**:2990−7.
43. McCormack JG, Denton RM. Intracellular calcium ions and intramitochondrial Ca^{2+} in the regulation of energy metabolism in mammalian tissues. *Proc Nutr Soc* 1990;**49**:57−75.
44. Pattullo MC, Cotter MA, Cameron NE, Barry JA. Effects of lengthened immobilization on functional and histochemical properties of rabbit tibialis anterior muscle. *Exp Physiol* 1992;**77**:433−42.
45. Gordon T, Pattullo MC. Plasticity of muscle fiber and motor unit types. *Exerc Sport Sci Rev* 1993;**21**:331−62.
46. Kokkonen J, Nelson AG, Eldredge C, Winchester JB. Chronic static stretching improves exercise performance. *Med Sci Sports Exerc* 2007;**39**:1825−31.
47. Gutin B, Wilkerson JE, Horvath SM, Rochelle RD. Physiologic response to endurance work as a function of prior exercise. *Int J Sports Med* 1981;**2**:87−91.